PHYSIOLOGICAL RESPONSE OF WALLEYE *(STIZOSTEDION VITREUM VITREUM)* TO HOOKING/PLAYING AND HOOKING/HOLDING STRESS

1988
ONTARIO FISHERIES
TECHNICAL REPORT SERIES
NO. 25

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Ontario
Ministry of
Natural
Resources

ISSN 0227-986X
ISBN 0-7729-3511-4

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Printed in Ontario, Canada

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Sobchuk, M. S., and N. Dawson. 1988. Physiological response of walleye (*Stizostedion vitreum vitreum*) to hooking/playing and hooking/holding stress. Ont. Fish. Tech. Rep. Ser. No. 25. 15 p.

This study evaluated the physiological response of walleye to hooking/playing and hooking/holding (on stringers) stress. Hooking/playing and hooking/holding stress were imposed under standardized conditions. Blood clotting time was used as an index of a non-specific physiological stress response. Plasma chloride and hematocrit were used to evaluate osmoregulatory changes and gill ion exchange function. Plasma glucose and plasma cholesterol were used to evaluate changes in carbohydrate and lipid metabolism. Both types of stress (hooking/playing and hooking/holding) produced significant changes in each of the blood parameters measured, but did not cause any mortality. Physiological responses during the recovery phase were particularly apparent in blood clotting time, hematocrit and plasma glucose.

Key words: physiological response, hooking/playing stress, hooking/holding stress, walleye, catch and release

INTRODUCTION

Access to remote lakes in northern Ontario often improves suddenly as a result of road networks created by expanding timber harvesting operations. As a consequence of improved access, a dramatic increase in angling pressure for walleye (*Stizostedion vitreum vitreum* (Mitchill)), the most sought-after game fish in the region (Bedi and Clifford 1982), often occurs. The high catch-per-unit-effort initially experienced on these lakes promotes the capture and release of large numbers of walleye since anglers realize that they can readily achieve a legal possession limit (six walleye). In these situations, many anglers use a catch and release strategy to obtain the largest walleye possible for inclusion in their limit.

It is also common practice for anglers to capture walleye and secure them on stringers suspended from the sides of their boats. As angling continues, larger walleye that are captured replace smaller individuals on the stringer; the latter being released. This process of 'sorting' often causes walleye to be held on stringers for many hours prior to release.

It is commonly assumed by anglers that the walleye they release will not suffer any significant mortality as a direct or indirect result of the capture experience. However, an immediate mortality factor associated with hooking, playing, handling and releasing fish has been demonstrated for rainbow trout (*Salmo gairdneri*) (Mason and Hunt 1967); cutthroat trout (*Salmo clarki*) (Marnell and Hunsaker 1970); lake trout (*Salvelinus namaycush*) (Falk et al. 1974); largemouth bass (*Micropterus salmoides*) (Pelzman 1978); and Atlantic salmon (*Salmo salar*) (Warner 1979). There have not been any studies conducted to quantify hooking and holding mortality for walleye.

In addition to immediate hook-induced mortality, fisheries managers must also be concerned with lethal and sublethal physiological responses caused by hooking, playing and handling stress. Parker and Black (1959) demonstrated a delayed mortality rate of 71% for troll-caught chinook salmon (*Oncorhynchus tshawytscha*). Mortality in that study was strongly correlated with elevated plasma lactate levels. Bouck and Ball (1966) demonstrated a high delayed mortality (87%, 3-10 days after the stressful event) among rainbow trout hooked on artificial lures and played until exhaustion. Mortality was correlated with changes in hemoglobin concentration, erythrocyte sizes, plasma protein concentrations and plasma protein fractions. May (1974) reported a delayed mortality of 15% for largemouth bass captured and released during a Bass Anglers Sportsmans Society (BASS)

tournament. Physiological responses were not quantified in that study.

Although hooking and playing stress may not always be lethal, it does cause significant metabolic and osmoregulatory disturbances (Mazeaud et al. 1977; Wydoski 1977) in fish and may increase their vulnerability to predation or susceptibility to disease during the recovery period (Wedemeyer 1970; Snieszko 1974).

Because walleye are often subjected to voluntary catch and release fishing, the study reported here was designed to evaluate (as measured by changes in blood chemistry) physiological response in walleye produced by hooking, playing and holding these fish under standardized conditions. Our use of the word stress is based on Brett's (1958) definition; "a state produced by any environmental or other factor which extends the adaptive responses of an animal beyond the normal range or which disturbs the normal functioning to such an extent that, in either case, the chances of survival are significantly reduced".

METHODS AND MATERIALS

The experiments were conducted between May 10 and May 18, 1984 at Albert Falls (50°23'N, 87°15'W) on the Kawashkagama River in the Geraldton District of the Ontario Ministry of Natural Resources (OMNR). Walleye used in these experiments varied in total length from 32.4 to 56.4 cm, and in total weight from 270 to 1650 gm. No distinction between male and female walleye was made in our experiments. Walleye in spawning condition were used in these experiments because it is in the spring, during or immediately after the spawning run, when they are subjected to the greatest catch and release fishing.

EVALUATION OF ACUTE PHYSIOLOGICAL RESPONSES TO STRESS

Evaluation of the severity of acute physiological stress caused by hooking/-playing and hooking/holding (on stringers) involved two experiments. In the first experiment (hooking/playing), walleye were caught using an unbaited 30 g artificial lure (trade name "Mr. Twister") with a no. 4 hook, and played for 0, 1, 2, 3 and 4 min (10 fish per time interval). All walleye were required to resist the rod pressure during playing. Walleye which became quiescent during playing were stimulated to resist rod pressure by raising the rod tip. Only walleye that were hooked in the jaws were used in the experiment. Blood samples were taken immediately after each walleye was landed at the conclusion of a playing interval.

Blood samples were taken by making an incision in the caudal peduncle and collecting 600 ul from the caudal vessel using pediatric blood collecting tubes (trade name "microtainer") which contained EDTA as an anti-coagulant. Each sample was immediately centrifuged at 2700 rpm and stored at -20°C until analysed later in the laboratory. Mean values for each interval were compared to the 0 min control using the Student's t test (Snedecor and Cochran 1968).

The 0 min controls for this experiment and the second experiment were established by landing each walleye within 15 sec after it was hooked and immediately taking a blood sample. The 0 min controls were considered pre-stress levels even though it is recognized that the capture of walleye, no matter how rapid, will cause some level of stress.

To evaluate acute physiological stress caused by hooking walleye and then holding them on stringers, a second experiment was conducted in which walleye were captured using the same gear as previously described and then held on stringers for 0, 1, 2, 3 and 4 hr (10 fish per time interval). Stringers were comprised of a series of metal clips, one being applied around the mandible of each walleye. All walleye were landed within 15 s of being hooked. At the conclusion of each holding interval, walleye were removed from the stringer and a blood sample taken and stored using the methods previously described. Mean values for each interval were compared to the 0 min control using the Student's t test (Snedecor and Cochran 1968).

EVALUATION OF PHYSIOLOGICAL RESPONSES TO STRESS DURING RECOVERY PERIOD

The severity of any physiological response to hooking/playing stress was evaluated by hooking and playing 40 walleye for 3 min each and then placing them in a recovery tank (described below) for 72 hr. Only five walleye were placed in a recovery tank compartment at any one time. Individual blood samples from subgroups of five fish were taken after 0, 1, 2, 4, 8, 24, 48 and 72 hr using the methods previously described. Mean values per interval were calculated and compared to the 0 minute control using the Student's t test (Snedecor and Cochran 1968).

In a second experiment, the severity of any physiological response to hooking/-holding stress was evaluated by hooking and landing 40 walleye within 15 s each, holding them on stringers for 1 hr and then placing them in a recovery tank (described below) for 72 hr. Only five walleye were held on a stringer at any time. Individual blood samples from subgroups of five fish were taken after 0, 1, 2, 4, 8, 24, 48 and 72 hr using the methods previously described. Mean values per interval were calculated and compared to the 0 minute control using the Student's t test (Snedecor and Cochran 1968).

The recovery tank used in these experiments contained 350 litres of water and was constructed of 190 mm plywood and measured 2.44 m x 0.61 m. The tank was lined with black polyethylene to eliminate any physiological effects a light substrate might have on the walleye. It was divided into six compartments (0.82 x 0.61 x 0.61 m) using wooden frames covered with nylon mesh. Water from Albert Falls was ducted through a 10.16 cm ABS plastic pipe to the tank by gravity feed. A perforated 10.16 cm ABS plastic pipe at the tank provided water flow to each compartment of the tank. A vertical standpipe (10.16 cm ABS pipe) at the rear of the tank maintained a water level of 54 cm. Water temperature and dissolved oxygen in the tank and river were recorded twice daily (1200 hr, 1800 hr). Water temperatures varied from 5°C to 6°C for the duration of the experiments. Dissolved oxygen varied from 11.6 to 11.9 mg L^{-1} during the experiments.

SAMPLE ANALYSIS

Each blood sample was assayed for clotting time, hematocrit, glucose, chloride and cholesterol by the methods described by Wedemeyer and Yasutake (1977). Blood clotting time was used as an index of a non-specific physiological stress response. Plasma chloride and hematocrit were used to evaluate osmoregulatory changes and gill-ion exchange function. Plasma glucose and plasma cholesterol were used to evaluate changes in carbohydrate and lipid metabolism.

RESULTS

ACUTE PHYSIOLOGICAL RESPONSES

Hooking/playing experiment

Mean blood clotting time declined significantly after 1 min of playing (Fig. 1A). Increasing playing time to 4 min produced only slightly greater decreases in blood clotting time, although the differences were still significantly less than the initial value (pre-stress level) at time 0. Conversely, mean hematocrit increased significantly after 1 min of playing (Fig. 1B) and remained significantly greater than the pre-stress level after 4 min.

A significant hyperglycemia developed after 1 min of playing (Fig. 2A) and remained significantly greater than the pre-stress level for all 4 min. The hyperglycemic condition showed a marked elevation after 2 min of playing then declined slightly and was followed by a further increase at 4 min. Similarly, a significant hyperchloremia developed after 2 min of playing and continued to increase in magnitude following each increase in playing time up to 4 min (Fig. 2B).

Mean plasma cholesterol values declined as playing time increased to 3 min (Fig. 3), but was only significantly lower than the pre-stress level after 3 min of playing time. The value after 4

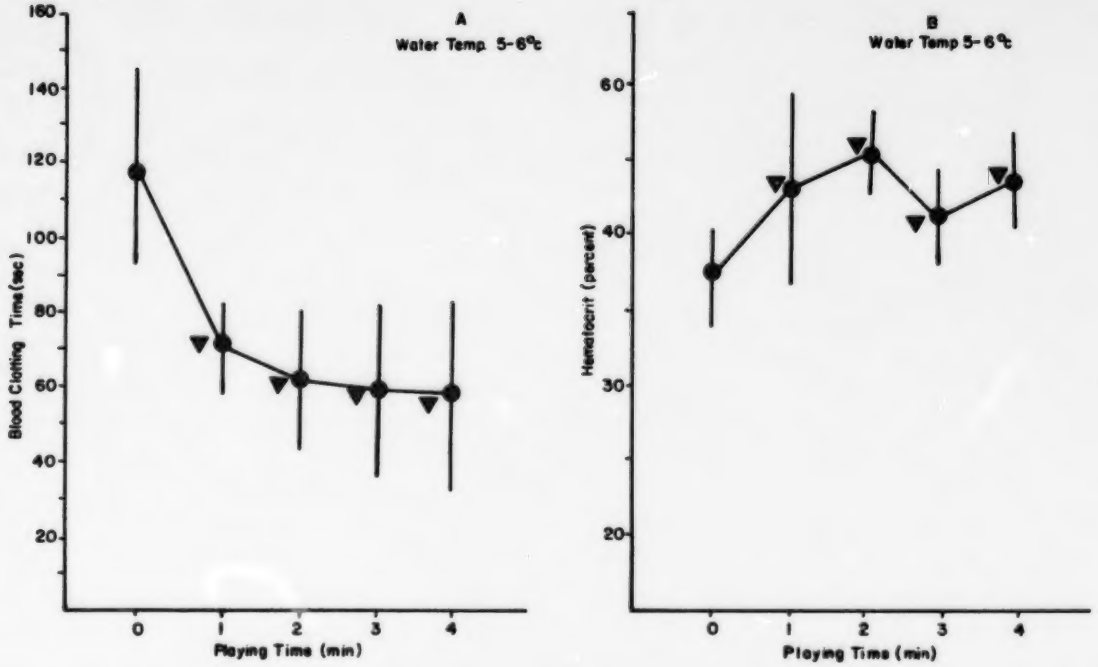

Fig. 1. Acute responses of blood clotting time (A) and hematocrit (B) for walleye following 0-4 min of hooking and playing stress at 5-6°C. Dots indicate the means and vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

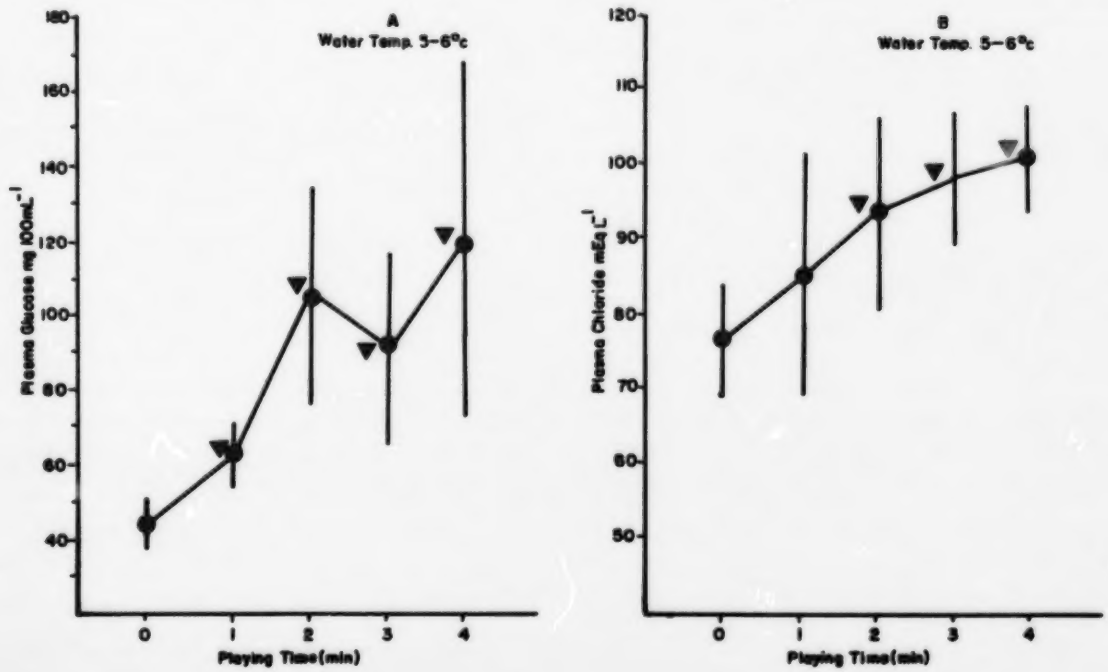

Fig. 2. Acute responses of plasma glucose (A) and plasma chloride (B) for walleye following 0-4 min of hooking and playing stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

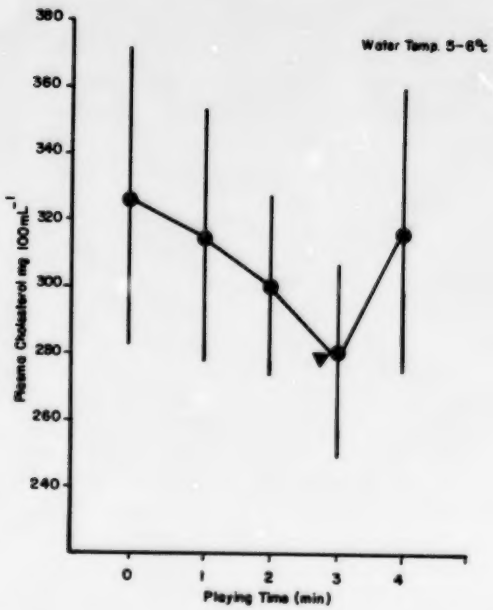

Fig. 3. Acute response of plasma cholesterol for walleye following 0-4 min of hooking and playing stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

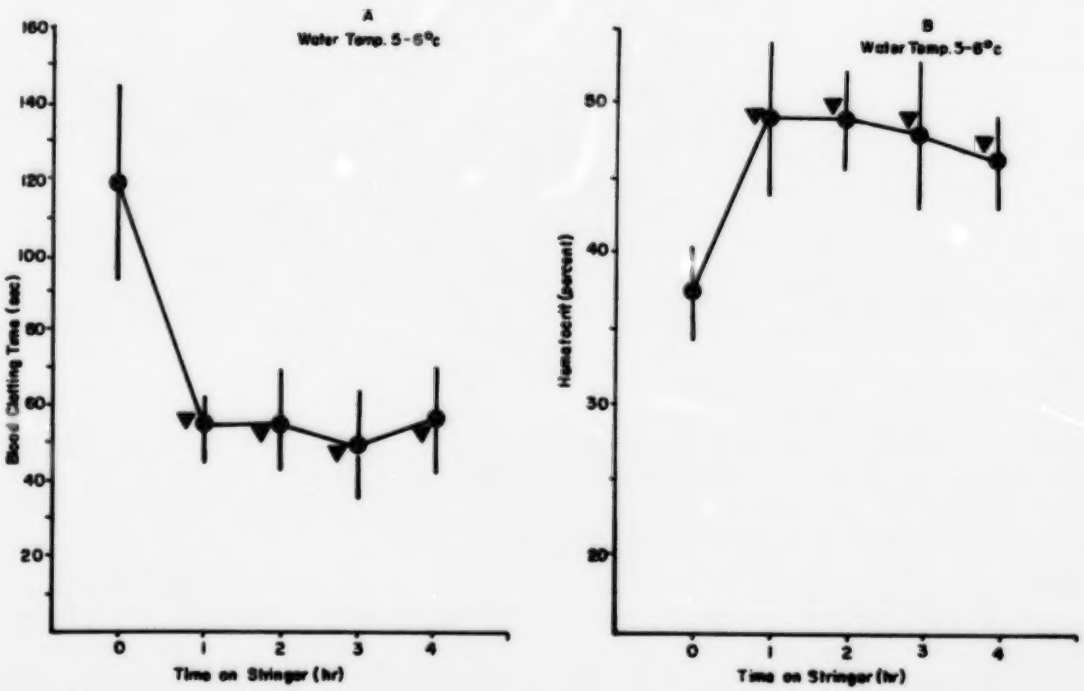

Fig. 4. Acute responses of blood clotting time (A) and hematocrit (B) for walleye following 0-4 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

min was not significantly different from the initial (pre-stress) level. No immediate mortality occurred during the hooking/playing experiment.

Hooking/holding experiment

In the hooking/holding experiment, mean blood clotting time declined significantly after 1 hr of holding on stringers (Fig. 4A) and remained significantly lower than the pre-stress level after 4 hr. A significant increase in mean hematocrit was noted after 1 hr on the stringers (Fig. 4B). Increasing the holding time to 4 hr produced slight decreases in mean hematocrit values, but they were still significantly greater than the pre-stress level at time 0.

A very significant hyperglycemia above the pre-stress level developed after 1 hr of holding on stringers and remained at significantly higher levels with increased holding times up to 4 hr (Fig. 5A). A significant hyperchloremia developed after 1 hr of holding on stringers (Fig. 5B). Fluctuations of mean plasma chloride values occurred with increases in holding time, but remained significantly greater than the pre-stress level for 3 hr.

Mean plasma cholesterol values demonstrated an increasing trend with increases in holding time (Fig. 6). A significant difference was not apparent until 3 hr of holding time. No immediate mortality was observed.

PHYSIOLOGICAL RESPONSES DURING RECOVERY PERIOD

Hooking/playing experiment

Hooking and playing walleye in spawning condition for 3 min produced a response in several blood parameters during the recovery period. Mean blood clotting time, after a slight increase at 1-2 hr declined until 8-24 hr (Fig. 7A). It then increased rapidly and was significantly greater than the pre-stress level by 72 hr. Mean hematocrit rose slightly during the recovery period for 24 hr then declined through 72 hr at which time it was significantly less than the pre-stress level (Fig. 7B).

Changes in plasma glucose levels were very pronounced. Walleye showed a significant hyperglycemia by 8 hr (Fig. 8A). At 24 hr, plasma glucose levels were significantly below the pre-stress level. Mean plasma chloride values demonstrated wide fluctuations during the recovery period, but were not significantly different from the pre-stress level even after 72 hr (Fig. 8B).

Mean plasma cholesterol values increased slightly for the first 4 hr of the recovery period (Fig. 9) and then exhibited an irregular but declining trend to 72 hr. None of the values was significantly different from the pre-stress level. No delayed mortality was observed.

Hooking/holding experiment

The response of mean blood clotting time to hooking and holding on stringers for 1 hr (Fig. 10A) was similar to that noted for hooking and playing (Fig. 7A). An initial, but non-significant increase was followed by a steady decline through 8 hr. After 24 hr, mean blood clotting time increased rapidly, but was only significantly greater than the pre-stress level at 72 hr. Mean hematocrit values had declined significantly from the initial value by 2 hr (Fig. 10B) and were still significantly less than the pre-stress level by 72 hr.

Plasma glucose values showed a marked increase after 2 hr (Fig. 11A). The elevated hyperglycemic condition was significantly greater than the pre-stress level at 8 hr then rapidly declined through 72 hr at which time it was significantly less than the pre-stress level. Mean plasma chloride values fluctuated widely during the recovery period, but did not differ significantly from the pre-stress level (Fig. 11B).

A decline in mean plasma cholesterol occurred after 1 hr of recovery time and continued through 8 hr (Fig. 12). A slight increase occurred at 24 hr and was followed by a significant decline at 48 hr. No delayed mortality was observed.

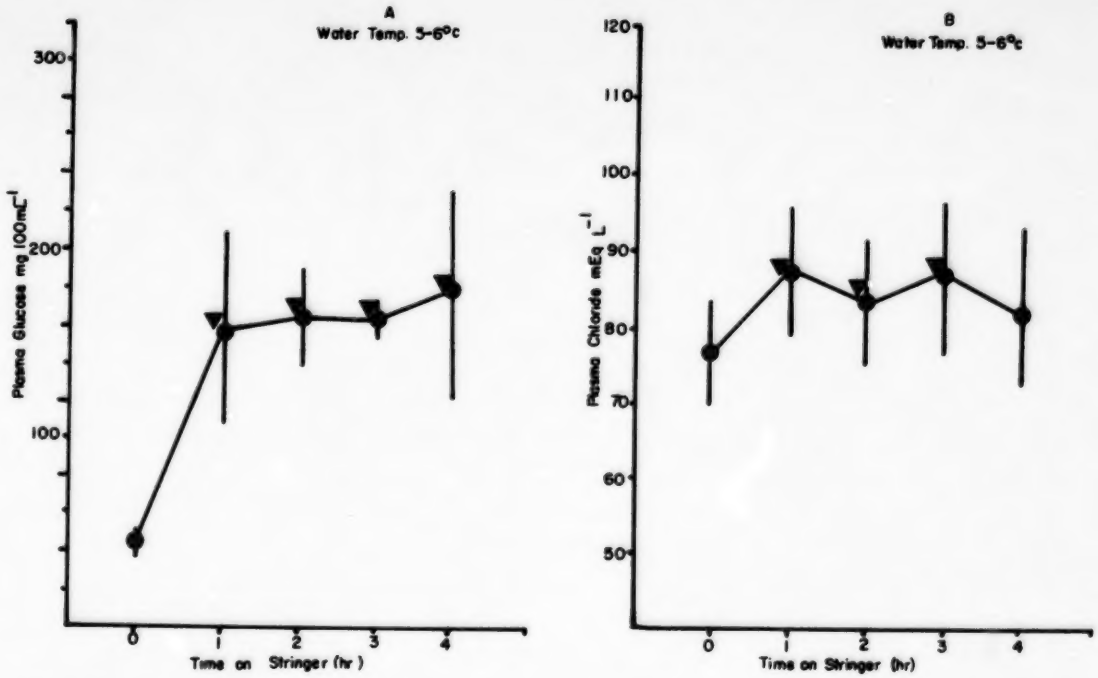

Fig. 5. Acute responses of plasma glucose (A) and plasma chloride (B) for walleye following 0-4 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

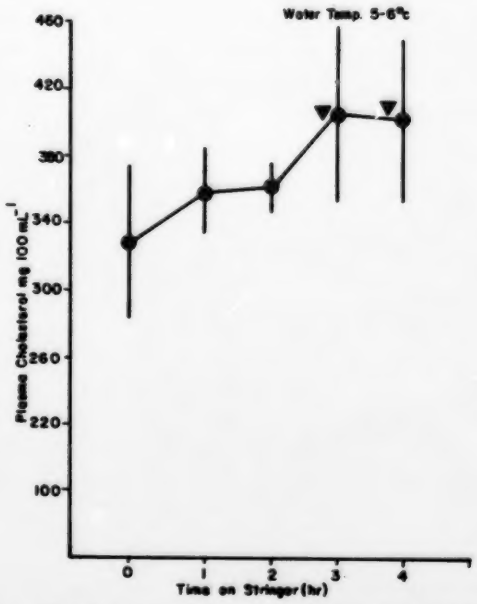

Fig. 6. Acute response of plasma cholesterol for walleye following 0-4 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 10 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

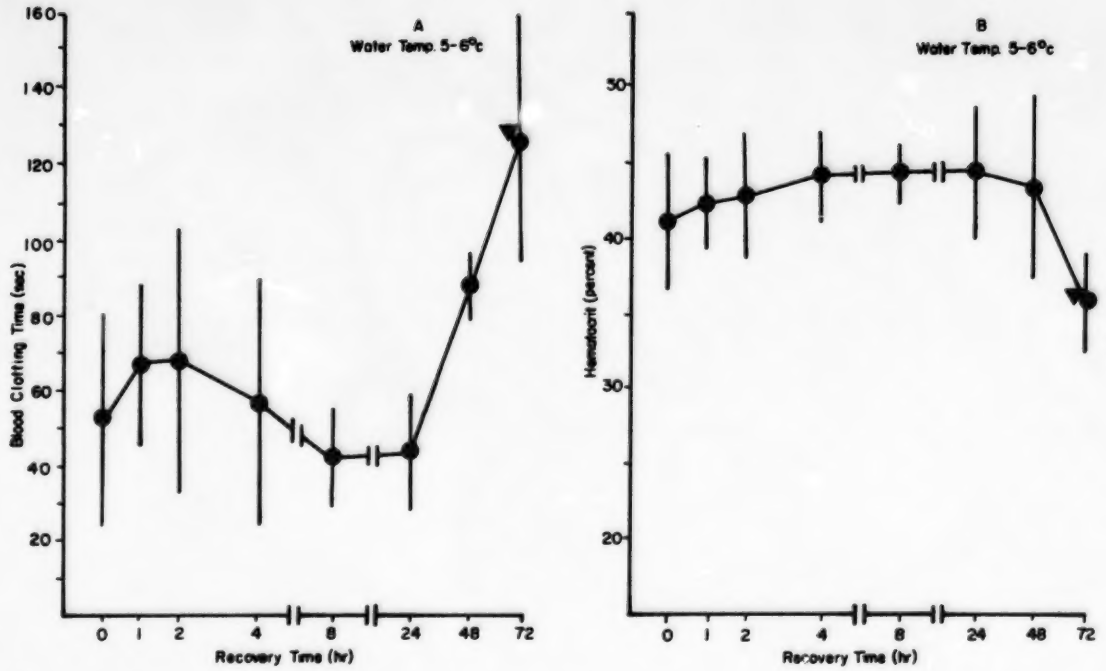

Fig. 7. Responses of blood clotting time (A) and hematocrit (B) during the recovery period for walleye subjected to 3 min of hooking and playing stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of 5 fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

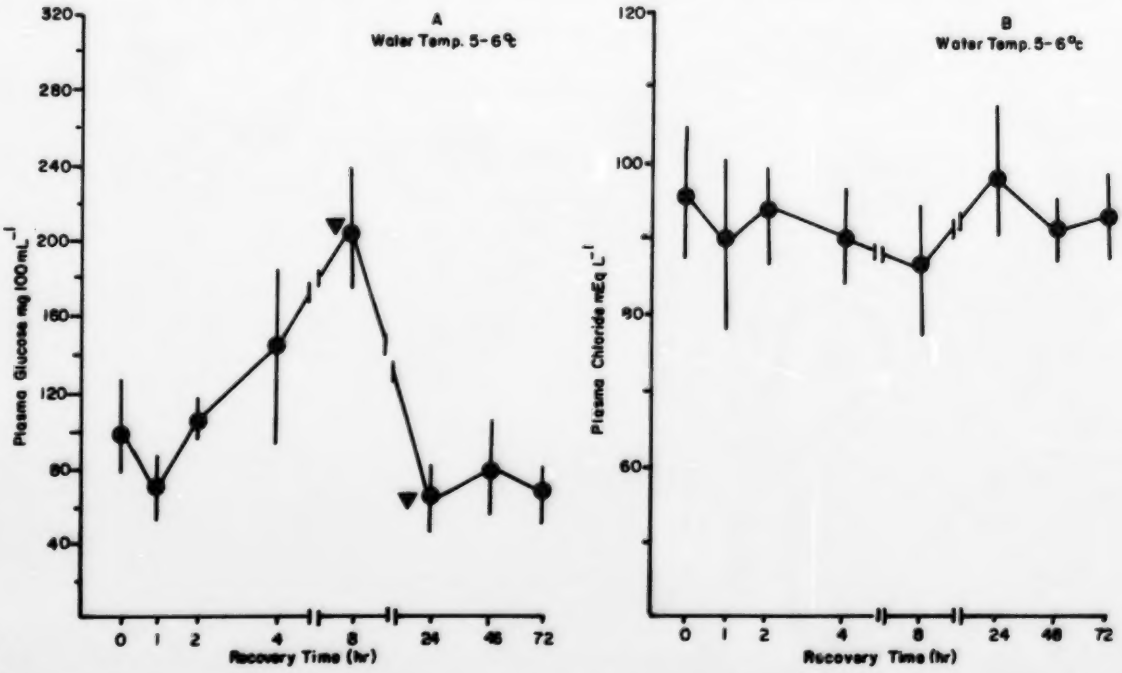

Fig. 8. Responses of plasma glucose (A) and plasma chloride (B) during the recovery period for walleye subjected to 3 min of hooking and playing stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of five fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

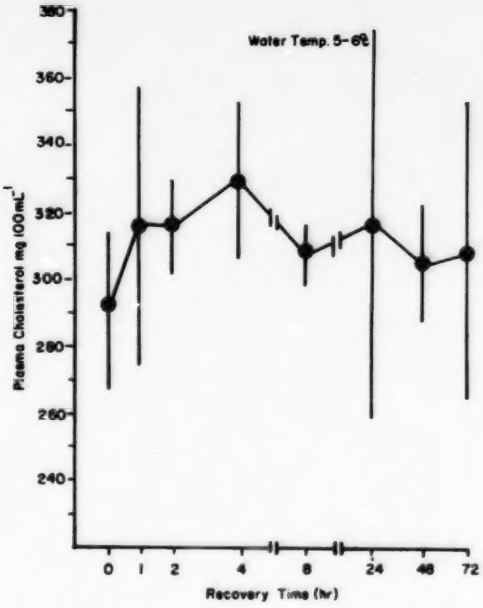

Fig. 9. Response of plasma cholesterol during the recovery period for walleye subjected to 3 min of hooking and playing stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of five fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

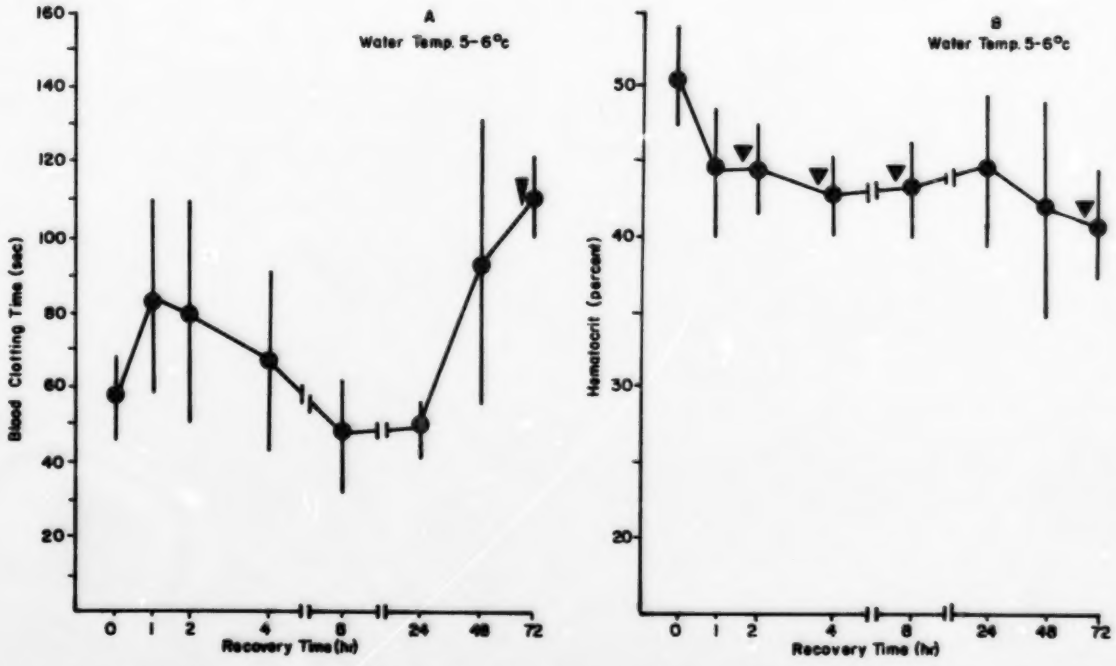

Fig. 10. Responses of blood clotting time (A) and hematocrit (B) during the recovery period for walleye subjected to 1 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of five fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

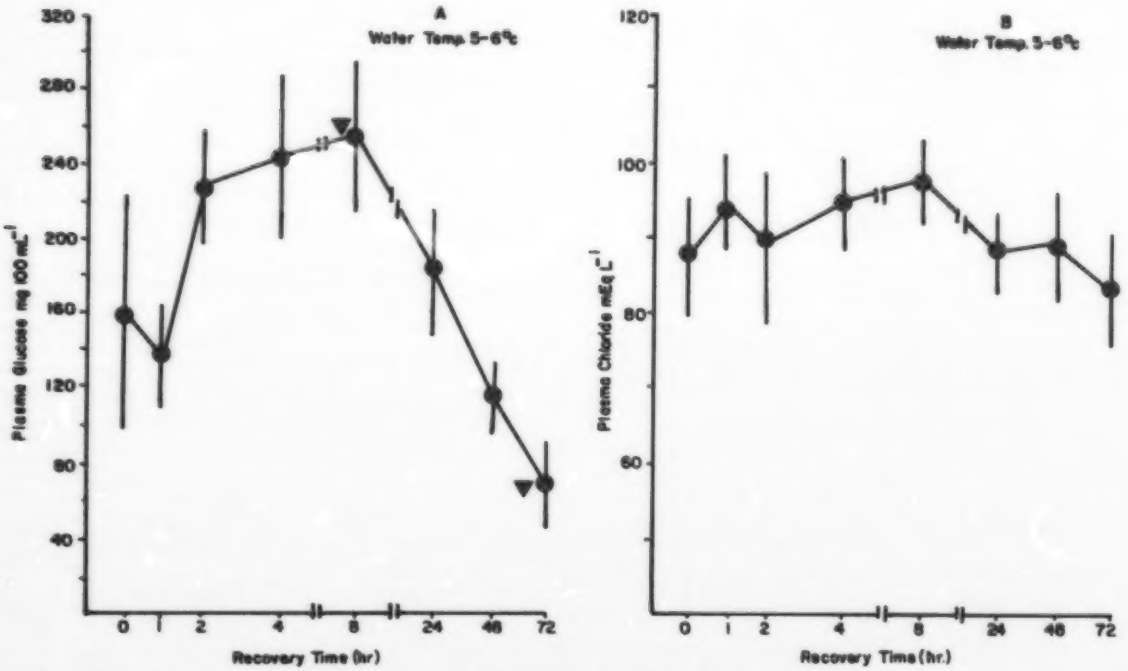

Fig. 11. Responses of plasma glucose (A) and plasma chloride (B) during the recovery period for walleye subjected to 1 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of five fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

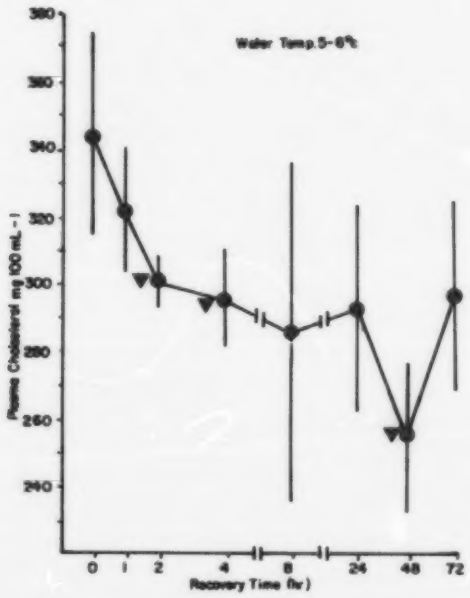

Fig. 12. Response of plasma cholesterol during the recovery period for walleye subjected to 1 hr of holding (on stringers) stress at 5-6°C. Dots indicate the means and the vertical lines the standard errors for groups of five fish. A ▼ indicates a significant difference (P <0.05) from the initial value (pre-stress level) at time 0.

DISCUSSION

ACUTE PHYSIOLOGICAL RESPONSES

Acute physiological changes, as measured by specific blood parameters, were apparent in walleye in spawning condition which were subjected to hooking/-playing and hooking/holding stress at water temperatures of 5-6°C. The significant declines in mean blood clotting time noted in the hooking/playing and hooking/holding experiments have also been demonstrated for wild and hatchery-reared rainbow trout subjected to hooking stress (Bouck and Ball 1966; Casillas and Smith 1977). Casillas and Smith (1977) have suggested that the blood coagulation system is involved in the general response to stress. Reduced blood clotting time may be related to alterations in the composition of plasma proteins (Bouck and Ball 1966).

Bouck and Ball (1966) have suggested that mortality of rainbow trout in their experiments was due to *in vivo* blood coagulation. High plasma lactate levels and sluggish circulation (Black 1958), which often accompany stress, could create an internal environment which could promote *in vivo* blood coagulation.

Elevated hematocrit values in response to hooking/playing and hooking/holding stress may be due to splenic contraction (Black 1955), erythrocyte swelling (Albers 1970), or the movement of water from blood to tissues (hemoconcentration) (Black et al. 1966). Miles et al. (1974) proposed that increases in hematocrit might be due to water loss through increased urine flow, similar to that observed by Hunn (1969) in rainbow trout.

One of the most pronounced secondary effects of stress in teleosts is the disturbance of carbohydrate metabolism as characterized by elevated plasma glucose levels (Mazeaud et al. 1977). The development of significant hyperglycemia apparent in these experiments has also been demonstrated in wild and hatchery-reared rainbow trout subjected to hooking and playing stress (Wydoski et al. 1976). Hyperglycemia related to the mode of capture, handling and transportation stress has been demonstrated in northern pike (*Esox lucius*) (MacKay and Beatty 1968) and muskellunge (*Esox masquinongy*) (Miles et al. 1974).

It appeared that hyperglycemia was more pronounced and of greater duration in larger walleye. The small sample size in our experiments prevented accurate quantification of this relationship. Wydoski et al. (1976) found that hyperglycemia was more severe and took longer to subside in large rainbow trout than in smaller individuals.

Changes in plasma glucose levels are due to the mobilization of muscle and liver glycogen stores by increased adrenal production of catecholamines and glucocorticoids mediated through stress (Nakano and Tomlinson 1967; Hill and Fromm 1968). Lower liver glycogen levels related to the physiological condition of individual fish may increase the risk of mortality if subjected to stress. Pasanen et al. (1979) noted significantly lower glycogen levels in female Baltic cisco (*Coregonus albula*) than in males in autumn and suggested that this was due to gonadal development in the females. The subsequent death of six females during a mark-recapture experiment was attributed to the exhaustion of liver glycogen stores.

Although not measured in these experiments, elevations in plasma lactate levels are usually associated with increases in plasma glucose levels (Farmer and Beamish 1969). Elevated plasma lactate levels have been suggested as a contributing factor in the deaths of rainbow trout (Bouck and Ball 1966), Pacific salmon (Parker et al. 1959) and chinook salmon (Parker and Black 1959). In walleye, elevated plasma lactate levels may become an important factor contributing to mortality as water temperatures increase during summer months.

The development of significant hyperchloremia in our experiments differs from results of stress experiments on various salmonid species. Houston et al. (1971) found that plasma chloride concentration fell during the immediate post-operative period in rainbow trout, but was followed by a slow increase back to control levels. Wydoski et al.

(1976) reported that the development of hypochloremia was a common consequence following hooking stress in rainbow trout. The development of hyperchloremia in our experiments may be due to increased chloride cell activity or changes in the reabsorption of electrolytes by kidney tubules.

Changes in sterol metabolism as measured by plasma cholesterol levels were also apparent in the hooking/playing and hooking/holding experiments. The progressive hypocholesterolemia noted in the hooking/playing experiment is similar to that noted by Wedemeyer (1973) in coho salmon (*Oncorhynchus kisutch*) subjected to a water temperature increase from 10°C to 20°C. The decrease in plasma cholesterol may be due to an increase in hepatic metabolism together with bilary secretion (Wedemeyer 1973). It is suggested that the development of a significant hypocholesterolemia may be enhanced at higher water temperatures due to temperature stimulated increases in hepatic metabolism.

The development of significant hypercholesterolemia in walleye subjected to hooking/holding stress may signify a two stage stress response. The stress of hooking and playing, even though of brief duration, may have caused a mobilization of cholesterol stores possibly stimulated by adrenal release of corticosteroids. The second stage of the stress response could involve an increase in hepatic metabolism as proposed above, but the quiescence of walleye during the holding experiment may have precluded development of this secondary response.

In general, the hooking/holding experiment produced somewhat surprising results in that the intensity of the blood parameter responses was only slightly greater than in the hooking/playing experiment, even though the level of applied stress was presumed to be greater. This approximation of results suggests that the experiment may not have been representative of actual, more severe, angler handling conditions, or that walleye have the physiological ability to compensate for a greater level of stress.

Observations of walleye when secured on the stringers showed that they remained quiescent during the stress periods. The fish were not disturbed while held on stringers as they would have been under angler holding conditions. Therefore, the response of walleye to hooking/holding is considered to be representative of baseline stringer stress levels only. Although there were no mortalities, the results emphasize the need for proper handling and release techniques, particularly in legislated catch and release programs.

The fact that there was no initial or delayed mortality in these experiments also suggests that walleye have the physiological ability to adapt to certain levels of stress. However, these results must not be interpreted to mean that catch and release situations are free of mortality. Rather, it is suggested that the fish used in these experiments were treated gently compared to typical treatment by anglers. Anglers, in many cases, would be releasing walleye which were hooked in the gills or esophagus. Walleye held on stringers are often towed through the water for extended periods and may be lifted out of the water several times. The rougher treatment by anglers could result in substantially higher levels of initial and delayed mortality due to more extensive physical injury and greater physiological responses. In addition, mortalities could result from post-hooking infections. Wedemeyer (1970) explained that the process of infection into disease is a strong function of the physiological state of the host, and that non-specific stresses operate to increase host susceptibility.

Greater susceptibility to predation, due to altered behaviour, reduced ability to escape or both, particularly in smaller individuals, is still another factor which could increase overall mortality. The possibility of increased mortality due to these factors suggests the need for further investigation of the impact of hooking/playing and hooking/holding stress.

PHYSIOLOGICAL RESPONSE DURING RECOVERY

The results of the recovery experiments indicate that stress of even a brief duration can cause immediate and

long lasting physiological responses in walleye. In some of the experiments, re-establishment of homeostasis (i.e. pre-stress levels) was not complete by 72 hr. Fish captured by means other than angling (e.g. trap nets) were not used as a control. Thus, the proportion of stress due to confinement and lack of food, and the extent to which these conditions affected recovery time, could not be determined.

The cold water (5-6°C) and high dissolved oxygen levels (11.6-11.9 mg L^{-1}) may have acted to reduce the level of the stress response and subsequent recovery period. Wydoski et al. (1976) found that the onset of changes in various haematological parameters was delayed several hours in cold water. Although there was no mortality in our study, several investigators (see for example Klein 1965; Hunsaker et al. 1970; Dotson 1982) have observed a general increase in hooking mortality associated with increasing water temperatures. Although mortality might have occurred if the experiments had been conducted at higher water temperatures, catch rates for walleye generally decline substantially following the spring fishery. Anglers are much less likely to practice voluntary catch and release fishing during the warmer months.

Given the cold water temperature and gentle treatment of the fish, we would not expect any mortality beyond 72 hr. Although Bouck and Ball (1966) reported a high delayed mortality (87%) in rainbow trout 3-10 days after capture, the fish were purposely played until exhaustion. Other studies in which the fish were held at least 10 days (Hunsaker et al. 1970; Marnell and Hunsaker 1970; Falk et al. 1974) found that most hooking mortality occurred within 48 hr.

The fact that walleye exhibit stress when hooked or retained may warrant the development of ethical procedures for releasing and holding these fish. Further work in this area should investigate the influence of water temperature, fish size, sex and the effects on fish in spawning condition on the physiological response pattern. It is also suggested that the assumptions of increased disease susceptibility and predation be examined more closely.

ACKNOWLEDGEMENTS

This study could not have been completed without the administrative expertise and encouragement of Bill Cook, who at the time of the study was Fish and Wildlife Supervisor in the Geraldton District. Special thanks go to Iris Jackson, Fish and Wildlife clerk, Geraldton District, for her diligence and hard work during the editing phases of the preparation of this paper. The authors are also grateful for the assistance Frank Comery, Fish and Wildlife technician, Geraldton District, provided during the field work.

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